VII. "On Stasis of the Blood, and Exudation." By Richard Norris, Esq. Communicated by Dr. Sharpey, Sec. R.S. Received August 28, 1862.

Confusion has been introduced into the question of stasis, as related to inflammation, by neglecting to discriminate between the various forms of stasis, of which there are four.

1. If the frog's web be exposed to certain irritants (*e.g.* chloroform), the arteries are so constricted that the heart-force is temporarily shut off from the capillaries, which become packed by the reflux of blood from the veins. This form of stasis is dissipated immediately on the cessation of the arterial constriction. The blueness of the extremities consequent upon exposure to cold is probably dependent on the same mechanism.

2. The second form of stasis depends upon such enfeeblement of the heart's force as interferes with the due propulsion of blood into the extreme vessels. It also disappears upon the re-establishment of a sufficient propulsive power.

3. The third form is that described by H. Weber as follows:— "If a limb [of a frog] be strangulated, there arises in its web within four to eight hours, without any irritation being applied, a stasis which is identical with inflammatory stasis, except that after sixty hours' duration it will be dissipated as soon as the circulation is set free." The removal of this stasis by the re-establishment of the circulation distinguishes it from inflammatory stasis, and shows its relation to the forms already described.

4. The fourth form of stasis is producible (artificially) by the application of irritants, and has for its specific characteristics—

α. It is readily induced when the heart-force is unimpaired and the blood-channels are free.

β. It requires hours or even days for its dissipation, or it may even be irresolvable.

γ. It presents under the microscope a homogeneous appearance, as if the vessels had been injected with a tinted size or gelatine. The outlines of the corpuscles are undistinguishable. I call this "homogeneous" or "inflammatory" stasis.

It is unnecessary to offer additional evidence upon the first

characteristic, which is an accepted dogma. The following experiment refers to the mode of resolution.

March 24th, 1861.—On examining the web of a frog which had been inflamed with tincture of iodine the day previously, I watched a vessel in which the homogeneous stasis existed, and observed the stasis to resolve in a peculiar manner. A normal current, such as is usually seen in capillaries, was circulating in the direction from A to B (fig. 1,) and impinging on the contents of the obstructed vessel C. The stagnation in the vessel C appeared to thaw as it were. The corpuscles were not pushed onwards in the mass, but seemed to take on the appearance of the impinging current, and the parts so reduced from the homogeneous to the heterogeneous condition did not appear to contain any great excess of corpuscles. This action soon extended through the whole length of the vessel, and immediately this was consummated a perfect current set in as in fig. 2. At the extreme edge of another division of this web I noticed two stagnated loops as in fig. 3. The circula-

Fig. 1. Fig. 2.

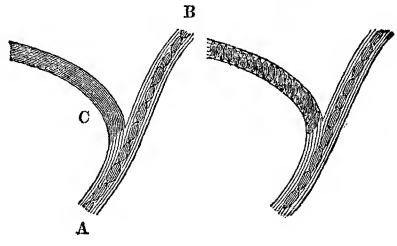

Fig. 3. Fig. 4.

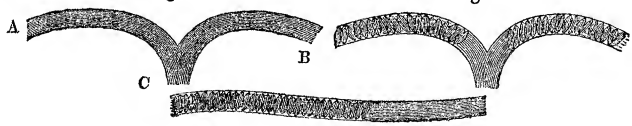

Fig. 5.

tion around them was in full activity. These I watched for more than an hour, and observed them to become gradually much lighter in colour, passing from a deep red to a pale orange. The point of junction C retained its depth of tint much the longest. All this time the contents of the vessels maintained perfectly their homogeneous character. At length all at once the outlines of the corpuscles became visible as in fig. 4, and the circulation was re-established. In this case, as in the previous one, there was no pushing on before of a plug of adherent corpuscles, but a gradual permeation of the liquor

sanguinis from the points A and B (fig. 3), with subsequent alteration of both the colour and disposition of the corpuscles. Fig. 5 also shows the progressive resolution of homogeneous stasis in a capillary vessel.

November 14th, 1861.—I took a vigorous frog, and having observed that the circulation was healthy, placed a ligature around a limb. On again observing I found the circulation arrested, but the corpuscles were very distinctly seen with $\frac{1}{4}$-inch power, and floated about with facility when the web was touched. I now applied to the web a small drop of chloroform, and, on again looking, found many of the capillaries had assumed the homogeneous condition. These observations strongly impressed me with the view that homogeneous stasis depends upon a new condition of the corpuscles, and not upon their crowding. The cause of homogeneous stasis appears to be the withdrawal of fluid from the capillaries, leading to a local modification of the liquor sanguinis, and consequently to the cohesion of the corpuscles with each other and the walls of the vessels, in obedience to the principles explained in my paper "On the Causes of various Phenomena of Attraction and Adhesion, as exhibited in Solid Bodies, Films, Vesicles, Liquid Globules, and Blood-corpuscles," in the present Number of the 'Proceedings.'

It is true that homogeneous stasis may occur in vessels previously packed with corpuscles, and we then find that the homogeneously solidified portion is of a much redder colour than when it occurs in a part previously free; in both cases irritation is essential to its production.

Again, corpuscular packing may arise as a secondary effect in consequence of the obstruction of the current by homogeneous stasis, the heart-force being unimpaired.

If the plug be pressed out of the cut extremity of a vessel obstructed by homogeneous stasis, we find the corpuscles adherent to each other; some have burst; we find also free nuclei.

In homogeneous stasis a certain quantity of colouring matter escapes from the corpuscles, and probably contributes to the appearance of homogeneity.

Stasis generally occurs first at the venous radicles, because here the *vis à tergo* is weaker.

Having attributed inflammatory stasis to a modification of the

liquor sanguinis, it is incumbent on me to show how and why this modification occurs.

Whilst it must be admitted that the cellular elements of the tissues have the power of imbibing and utilizing fluid plasma when it is brought into immediate apposition to them, there is not convincing evidence that these elements exercise any positive educing force upon this fluid while it remains within the vessels. Still less is there evidence that, of two cells situated the one nearer to, the other more remote from the vessel, the latter has any attractive power superior to that of the former, which, to secure its nutrition on the theory of positive attraction, it must have, for the plasma attracted by the nearer cell would remain in the possession of that cell till removed from it by a superior force.

The law of diosmosis suffices to explain the supply of fluid plasma to the cellular elements without recurrence to the hypothesis of a positive attractive force resident in the cells themselves. It is impossible to doubt that such structures as capillaries are diosmotic.

The more braced the condition of the minute vessels the less diosmosis, and *vice versâ*. It is not during contraction of the minute vessels produced by irritants that stasis occurs, but during the relaxation consequent on such contraction,—a relaxation which must be attributed to exhaustion of their irritability by the stimulus applied. This relaxation permits the diosmotic escape of fluid from the vessels, causing an inspissation of the plasma within them, and consequent adhesion of the corpuscles constituting inflammatory stasis. This escape of fluid may be termed primary exudation.

The muscular paralysis in question is not necessarily connected with neural paralysis, since it is producible in parts which, though abounding in contractile elements, are without nervous tissue, as, for example, in the umbilical cord. In fact the more completely the nervous influence is removed and destroyed the more sensitive does the muscular tissue become to irritants.

Neural paralysis does undoubtedly play a part in inflammation. Whilst the nerve-influence is exercised over a part, it affords a protective influence which renders the contractile elements less sensitive to local irritants, and consequently less prone to that absolute muscular paralysis which precedes primary exudation. But the neural paralysis does not necessarily involve absolute muscular paralysis,

although it facilitates its production. In corroboration of these views I may refer to the well-known results following the section of various nerves, *e. g.* the pneumogastric and the fifth.

This muscular paralysis is probably producible directly by muscle-sedatives, as it is indirectly by muscle-irritants. Since diminished nerve-force produces hyperæmia, and since diminished nerve-force furnishes the conditions under which homogeneous, *i. e.* inflammatory, stasis is most prone to occur, we see why hyperæmia and inflammation are so frequently conjoined. The experiments of Claude Bernard on the sympathetic, while showing the connexion between neural paralysis and hyperæmia, also indicate that neither neural paralysis nor hyperæmia are convertible terms with inflammation.

A distinction is drawn between that diosmotic exudation which leads to homogeneous stasis, and that subsequent copious transudation of fluid which fills up the interstices of tissues or leaks into cavities.

If in a frog's web homogeneous stasis has occurred in the venous radicles so as to completely prevent the passage of the blood into the veins, the current in the capillaries and supplying arteries might naturally be expected to be brought to a stand, as it certainly would be if the walls of the capillaries removed from the immediate seat of the obstruction were impervious ; but so far from this being the case, the blood brought to the part is seen to pass on in a perfectly regular manner without the slightest rebound. This absence of rebound is an evidence that the liquor sanguinis is passing through the vascular parietes at the same rate it is being propelled into the obstructed vessels.

It is not till the capillaries become packed with corpuscles and the circulation is confined to the arterial trunk that any rebound after the ventricular contraction becomes apparent. This rebound is the cause of throbbing in inflamed parts.

The views here briefly given seem to me to form a consistent theory, supported by experiment as far as the subject admits of experiment, in accordance with the phenomena of inflammation as observed in the warm-blooded animals.

Fig. 1.

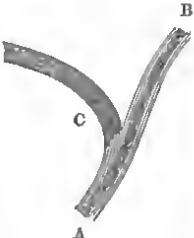

Fig. 2.

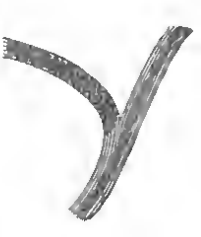

Fig. 3.

Fig. 4.

Fig. 5.